into which the nozzle of a hand spray-producer projects. The retort of the spray contains the infective mixture, and the operator is separated from the spray by a long India-rubber tube. If necessary, he may shield himself by means of a respirator; but, as a rule, the box can be placed in the open air and at such a distance away that he is in no danger.

Spore-bearing organisms may be used in the form of dust, which may be either blown into the vessel containing the animal or placed on the floor of the vessel, which is then shaken to raise the dust.

G. Infection by the Food.—This mode of experimentation is used to test the possibility of infection from the intestine, and in carrying it out there are two points to be attended to: care must be taken, first, that infection does not occur from the mouth or throat; and, secondly, that the infective material is not destroyed in the stomach. At first the experiments were made by simply mixing the infective material with the food, but in these cases infection often occurred from the mouth and throat. To avoid this, Koch adopted the plan of taking two small slices of boiled potato, scooping out a small depression in each piece, filling this depression with the bacteria, placing the two slices in apposition so that the bacteria were enclosed in the potato, and pushing the piece of potato to the back of the tongue so that it was swallowed at once without being chewed.

As regards the second danger, it is only present in the case of non-spore-bearing bacteria. Spores are able to resist the action of the gastric juice, whereas adult forms are, as a rule, destroyed by it. In the case of the cholera bacillus, Koch has devised a plan in the case of guinea-pigs by which the bacilli escape the action of the gastric juice. He first neutralizes the contents of the stomach by introducing into it eighty minims of a five per cent. solution of carbonate of soda. Ten minutes later a culture of the cholera bacillus is injected through a stomach tube (catheter). Lest the bacteria, which thus escape the action of the gastric juice and enter the intestine alive, should pass too rapidly through the small intestine, he then injects a solution of opium into the abdominal cavity (sixteen minims of the tincture for every six ounces of the weight of the animal). Koch hopes that this plan will prove useful in testing other forms of bacteria.

THE ETIOLOGY AND MECHANISM OF ASTHMA.

By William C. Glasgow, M.D.,

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No subject in medicine has given rise to greater discussion or criticism than that included in the term "Asthma." It was indiscriminately applied by the older writers to dyspnœa arising from various causes, and at the present time, although the majority confine the term to paroxysmal dyspnœa, accompanied by characteristic physical signs, still we find it used in a loose way to describe dyspnœa due to entirely different pathological conditions.

In this article I wish to confine the term to the paroxysmal dyspnœa which is always characterized by positive physical signs. I will exclude from consideration all other forms. Cardiac asthma due to a weakening of the heart, is not asthma; nor can we include the smothering of pure emphysema, although emphysema may be complicated with asthma. Suffocative attacks of bronchitis belong to the latter disease, although asthma may be also engrafted upon it and immeasurably increase the distress of the sufferer. In various diseases of the nervous system there is marked and paroxysmal dyspnœa, but the physical signs of asthma are always absent.

The older writers divided asthma into idiopathic or essential, and reflex. In the class of idiopathic asthmas were included all for which no reflex origin could be discovered; whilst in those classed as reflex, the point of irritation was mainly found in the bronchi and the mucous membrane of the gastro-enteric canal, the uterus, or the uterine appendages. The number of reflex cases was limited, and the large majority of cases were classified as idiopathic.

Modern investigations have proven that asthma of reflex origin is the most common, and at the present day it seems not improbable that those cases for which no local point of irritation can be discovered, and which seem idiopathic in character, will, by later investigators, be brought into the reflex class. Emotional asthma seems to be most certainly of the essential character; but when we consider it as analogous to vasomotor disturbances of the skin seen in emotional states, causing blushing or pallor, we may find a reflex origin through some of the senses.

Voltalini first drew attention to asthma dependent on the presence of nasal polypi, and showed that the removal of the polypi was followed by a cessation of the asthma. Later investigations have proven that it is not the polypi alone, but rather the peculiar condition of the nasal chamber in which the polypi are found that is the immediate cause of the asthmatic attack. Hack and John Mackenzie, of Baltimore, have especially drawn attention to the fact that a condition of congestion is at times present, and that the cavernous tissues of the turbinated bones become hyperæsthetic. They have endeavored to prove that the special sensitive areas are found either at the anterior or posterior extremities of the turbinated bones. It has now, however, been proven that the whole surface of the membrane is involved more or less in this sensitive area. In this sensitive condition, the presence of an irritant is capable of producing the asthmatic paroxysm. A polypus touching the sensitive area, retained secretions, dust, intense light, or anything capable of producing an impression on the nasal mucous membrane will provoke the attack. Even a swollen condition of the mucous membrane allowing a lateral pressure between the septum and the cavernous bodies, seems to be a provoking cause. That a nasal polypus alone is not capable of producing asthma, is shown by a case in which I removed a polypus from the lower turbinated bone with complete cessation of the asthmatic symptoms, although the upper portion of the chamber was still filled with nasal polypi. The asthma subsequently returned on a recurrence of the lower polypus.

In the pharynx we find an immediate cause in the presence of hyperæsthetic, enlarged follicles. In a case of this character the asthma was modified by an application to the group of follicles, and disappeared after the destruction of the same.

In 1882 I presented to the American Laryngological Society a paper termed "Laryngeal Asthma." In this article I sought to prove the connection between a hyperæmic or inflamed condition of the larynx and the asthmatic paroxysm. I cited several cases where the asthmatic paroxysm had been modified or entirely removed by the application of an anæsthetic or astringent solution to the larynx. The sensitive area is especially the inter-arytenoidal space and the membrane over the arytenoid cartilages. Since then I have seen a large number of these cases, and will cite the following as illustrative:

Through a misplacement of bottles, I pencilled the larynx of a man suffering with a severe asthmatic paroxysm, with a fifty per cent. solution of carbolic acid. The application caused intense burning, and the patient was able to swallow with great difficulty for several days. The asthmatic paroxysm, however, *was instantly broken on the application of the brush*, and since then he has been entirely free from asthma. I could report many cases in which the spasm has been instantly modified or entirely broken by the application to the larynx of a mild solution of carbolized iodine, or the insufflation of morphia powder. From the success of this form of treatment, the immediate and at times almost instantaneous relief given, I feel justified in saying that the sensitive area of the larynx, in a large number of cases, is the site of localized irritation provoking the asthmatic attack. From a study of a large number of asthmatics, I am fully convinced that the local site of irritation in the greater number lies in the upper air-passages. Störck, Nothnagel, Longet, and Schiff, have demonstrated that the sensitive cough area is located in the inter-arytenoidal space, the posterior surface of the trachea, and at the bifurcation of the bronchi. Later investigators (Hack, Haring, John Mackenzie, Daly, and Roe) have added to these the surface of the lower and middle turbinated bones.

Many cases of so-called bronchial asthma have their site of irritation in the nares, in the larynx, and probably in the posterior surface of the trachea, and the bifurcation of the bronchi. Cases of asthma caused by odors, the effluvia of animals, and the smell of flowers, or light, are due probably to a pathological condition of the nares. I am also inclined to believe that asthma due to emotional or psychical influence may in certain cases be due to the same cause. It is fully recognized that the so-called vasomotor catarrh, with turgescence and hyperæmia of the cavernous bodies, may be produced by mental impressions, the rose experiments of Dr. John Mackenzie being a striking example. It is also a well-known fact to laryngologists that a transient flushing or blushing of the mucous membrane takes place when the light of the reflector is thrown upon it. The following case is interesting as showing the dependence of a changed condition of the cavernous bodies on a psychical influence. In a patient of the St. Louis Post-Graduate Polyclinic the cavernous tissue of the nose was greatly swollen. I proposed to excise a portion of it. She demurred strongly, and when I insisted, became very much frightened: on looking again, I was astonished to find that the swollen tissue had become much paler, and had been reduced in size fully one-half. This was fully observed by members of the class.

Uterine asthma and peptic asthma, on which so much stress has been laid by Hyde Salter, may also have a possible connection with a certain condition of the upper air-passages. The coincidence of paroxysms of nervous dyspnœa with certain conditions of the uterus and its appendages, is well authenticated; but whether these paroxysms are real attacks of asthma or spells of nervous dyspnœa, in which we find rapid breathing with great intensity of the respiratory sounds, but no asthmatic râles, has not been so conclusively established.

Hyde Salter relates one case, reported by another physician, in which real asthmatic attacks occurred during pregnancy; and Engelmann, of St. Louis, has noted a case in which asthmatic attacks followed a retroflexed position of the uterus, and disappeared with the reposition of the organ; but in this case there is no record of the presence of asthmatic râles. Paroxysmal nervous dyspnœa is quite a frequent accompaniment of a disturbed condition of the nervous system. It is seen in hysteria, neurasthenia, and spinal irritation; it is seen in connection with disorders of the uterus or its appendages, and also in cases of impoverishment of the blood or blood-poisoning. Resembling true asthma in its paroxysmal attacks, it is, however, a distinctly different disorder. The dyspnœa is rather a rapid panting than the labored breathing of asthma; the respiratory sounds are simply intensified normal sounds, and the asthmatic râles are absent.

That asthma occurs at many of the critical periods of female life is certainly true. In regard to pregnancy, I have seen two cases in which the asthma disappeared with conception, and has not since returned.

Both of these patients were asthmatics of long standing. It is in these cases of uterine asthma that we can find often a possible nasal origin for the attacks. The connection between the condition of the cavernous bodies and uterine changes is very marked. Many women at the menstrual epoch have vasomotor catarrh, with great turgescence of the cavernous bodies and the mucous membrane. This condition in itself has been shown to be a sufficient cause for the attack, irrespective of the pathological condition of the uterus. The peptic asthma, on which so much stress is laid by Hyde Salter, may be fully explained by referring the irritation to the larynx. Gastric pharyngitis and gastric laryngitis are recognized forms of throat disease. They are dependent upon functional gastric disturbance, with the production of fermentation and acid secretions. In the peptic asthma quoted by Hyde Salter, the paroxysms occurred during the end of the digestive process, showing that the mere presence of food is not sufficient to cause the attack. The majority of asthmatics are dyspeptics, and it is possible that these cases were subject to a gastric laryngitis, in which the sensitive area of the larynx was involved. A similar condition is found to exist in so-called stomach cough. Although the local seat of irritation seems to be, in the majority of cases, in the upper air-passages, it is probable that a disordered condition of other organs exerts a positive influence in provoking the attack. As this is true in other nerve disorders, as migraine, epilepsy, and neuralgia, it is equally probable in asthma. For example, in a person with a disposition to asthma, the attacks may only attain the degree of the first stage, and give rise to scarcely noticeable symptoms. When, however, an increased irritation is added, as would occur through a disorder of the gastro-enteric canal or uterine disease, this added element would be sufficient to increase the irritation, and the first stage would grow into the second with the appearance of all the well-known asthmatic symptoms. A similar condition is found in neurotic palpitations occurring in a heart weakened by anæmia, where a torpid liver would provoke a disturbance, which, in a normal condition of the secretions, would be absent.

All writers on asthma have noticed the great difference in the severity of the paroxysms not only in different individuals, but even in the same individual at different times. Walsh has even assumed two forms of the disorder, one seemingly dependent on a spasmodic and the other on a paralytic condition of the bronchial muscles. If the asthmatic be closely studied during the paroxysm, we shall find a positive and characteristic connection existing between the severity of the attack and certain physical signs in the lungs. We can clearly divide the paroxysms into three stages, each stage characterized by positive physical signs, and with each stage a certain increasing degree of the severity of the symptom is noted.

The first stage may be called the state of

INSPIRATORY DYSPNŒA.

On examination we find the percussion sound unaltered; on auscultation the *inspiratory sound of the vesicular murmur is replaced by a high-pitched, prolonged blowing sound.* The expiratory sound may be absent or unchanged. This blowing inspiration is evidently *a blowing râle,* and it is caused by a cylindrical narrowing of the lumen of the bronchi, not sufficient to cause the sibilant or whistling râles. It is pathognomonic of the first stage of uncomplicated asthma, and differs from the rough inspiratory murmur of bronchitis or the intensified inspiration of the exaggerated respiration such as is heard in compensatory breathing or in the nervous dyspnœa of hysteria or blood poisoning.

When asthma is complicated with bronchitis, this inspiratory blowing sound loses, in a measure, its distinctive quality and becomes rougher and of lower pitch. When emphysema is present, the blowing quality of the sound is less marked, and it becomes much weakened and in some cases is absent.

The symptoms of the first stage are often slight; at times wanting. In some a slight tightness of the chest is experienced; in others a slight dyspnœa is felt on exertion; a paroxysmal cough most marked in the early morning hours is sometimes the only symptom.

The second stage may be called the stage of

EXPIRATORY DYSPNŒA.

It presents the physical signs usually recognized as characteristic of the asthmatic paroxysm. Percussion gives an increased resonance. On auscultation, a prolonged expiration is heard. Both expiration and inspiration may be covered by the sibilant and sonorous râles. In certain cases small moist râles of a liquid character are heard over limited portions of the chest. The symptoms of the second stage are those usually associated with the asthmatic paroxysms; the sense of oppression, the dyspnœa, and the slow, labored breathing are marked. The thorax heaves with each inspiration and the accessory muscles of respiration are brought into action, the patient is unable to lie down, but sits or moves in a fixed position, with raised shoulders and head projected forward. The face is livid and the alæ nasi dilated.

The third stage may be called the

STAGE OF ORTHOPNŒA.

Percussion gives a deep, drum-like resonance, showing the extreme distention of the vesicles. *On auscultation a short inspiratory whiff is heard, and the expiratory sound is inaudible.* The inspiratory whiff is most apparent over the larger bronchi. The symptoms of the third stage of asthma present a picture of the most supreme distress and agony. The patient stands with the hands resting on some fixed point of support, and the entire energy of the body is given up to the struggle for air. The true respiratory muscles seem to be completely inactive, and breathing is carried on entirely by the accessory muscles of respiration. The thorax is fully distended with little perceptible movement; the face becomes dusky, covered with perspiration—in strong contrast with the cold extremities; a sense of constriction is experienced around the body over the diaphragm.

These three stages are evidently produced by the varying and gradually lessening calibre of the bronchial tubes. In the first stage the calibre of the tubes is not sufficiently narrowed to produce the whistling râles. The inspiratory blowing râle heard in this stage is due to the moderate narrowing of the bronchi. A normal inspiration is the result of atmospheric pressure; the thorax is enlarged by the action of the thoracic muscles and the diaphragm, and the lungs are distended by the atmospheric pressure. The expiration is mainly performed by the lung vesicles. In the first stage the calibre of the bronchi is narrowed, but the walls of the vesicles remain intact and are normal: hence the expiratory sound is normal, but the inspiration assumes the peculiar high-pitched blowing found in this stage. In the second stage portions of the bronchi are narrowed to such a degree that the respiratory sounds assume the form of râles—the sonorous and whistling. The vesicles are distended through imperfect ventilation of the lungs and the energy of the stretched, elastic fibres is impaired. The result of this is a prolonged and labored expiration, in which the expiratory sound is formed by the sonorous and sibilant râles. In the third stage the lumen of the bronchi is reduced to a minute canal. The vesicles are distended to their utmost capacity; the bronchial mucous membrane is greatly swollen, and the diaphragm is either in a state of spasm or fixed and crowded by the over-distended lung. The greater portion of the air within the lung is residual air and there is no current except in the bronchi of larger calibre; aëration is performed slowly by the interchange of gases in this portion of the lung. There is no action of the vesicles or the diaphragm, and no expiration is heard; the inspiratory sound is produced by the passage of air through the glottis and larger bronchi. In some cases we can also see transition periods in which the first stage is verging into the second—shown by the presence of inspiration commencing with a blowing râle and ending in a sibilant; or with a blowing râle on inspiration and a sibilant or sonorous râle on expiration. The transition of the second to the third stage is seen in shortened expiratory râles which disappear completely as it lapses into the full third stage.

These different stages of asthma occur in the same individual at different times, although a certain similarity of type is apt to occur in different individuals. That they are simply stages of the same process, and that the varying physical sounds are all due to a narrowing of the bronchi, are fully proven by the effects of therapeutic efforts. For example, the inhalation of nitrite of amyl, or an application of carbolate of iodine to the larynx, will often instantly change the inspiratory whiff of the third stage to the prolonged expiration with the sibilant and sonorous râles of the second. This change is at once realized by the patient in a loosening of the constriction of the chest, and greater comfort and freedom of breathing. We can also, through the same means, produce a change from the whistling râles of the labored expiration of the second stage to the blowing râles of the first. When the asthmatic is subjected to some additional source of irritation, we find the increased distress of the patient explained by the change from the first to the second stage, or the second to the third. Although the asthmatic paroxysm is essentially due to a bronchial obstruction, we find it quite frequently interchangeable, and at times complicated with spasm of the glottis and spasm of the diaphragm. In one case, of a lady sixty years of age, I have seen a pure asthmatic spasm subside and followed in a few days by a distinct spasm of the glottis, with entire absence of asthmatic breathing; and again, in her next attack, glottic and bronchial spasm were absent, and spasm of the diaphragm was present. At this time a feeling of intense constriction was felt over the diaphragm, and the breathing was almost entirely superior costal, with a complete absence of the asthmatic râles. The sense of constriction in the upper part of the chest seen in the asthmatic, was wanting. In another case I have seen a true asthmatic paroxysm complicated, and at times synchronous, with spasm of the glottis. In this case the asthma disappeared, but the man died suddenly from glottic spasm. These conditions cannot be considered in reality as part of asthma, although often complicating it. They bear the same relation to the asthmatic paroxysm as exists in many other nerve disorders. For example, migraine, epilepsy, neuralgia, and insanity, are all interchangeable with asthma, and in families showing a disposition to nerve disorders they may be found in different individuals. That bronchitis and emphysema ultimately arise as sequelæ of asthma, is clearly probable, as the continued disturbance of the normal condition of the bronchial mucous membrane is apt to produce and incite pathological changes; and the great and frequent disturbance of the elastic fibres of the vesicle can only result in weakening and loss of tone. The right ventricle must also frequently suffer, undergoing dilatation and hypertrophy, as the great distention of the walls of the vesicles must seriously interfere with the normal pulmonary circulation.

The mechanism by which the asthmatic paroxysm is caused, has always been the subject of dispute and controversy. Laennec, followed by Andral, ascribed it to spasm of the circular fibres of the bronchi, causing cylindrical narrowing of these tubes. This may be said to be the prevailing doctrine at the present day, and it has been endorsed by many of the most eminent authorities in medicine; among these may be mentioned the names of C. J. B. Williams, Bergson, Hyde Salter, Trousseau, and Biermer. Rieseisen having demonstrated the existence of the circular fibres of the bronchi, others have sought to prove their capability of contraction under certain stimuli. Longet and Volkmann especially claim to have accomplished this by means of electro-stimulation of the vagus, but this result has not been produced when the experiments have been carried out by others. The theory of bronchial spasm as an element of the asthmatic paroxysm, has been warmly disputed. Budd rejected the idea, and he has been followed by Wintrich, Bamberger, Germain Sée, and others. Duchenne having demonstrated that faradization of the phrenic nerve caused spasm of the diaphragm, was inclined to consider this as an essential element of the paroxysm. In this he was supported by Wintrich and Bamberger. Traube denied the nervous origin of asthma, and ascribed the dyspnœa to the fluctionary hyperæmia of the bronchial mucous membrane. He supposed certain persons to have a certain predisposition to asthma, and that the predisposition consisted in a deficiency of the elastic fibre in the lung, allowing a ready distention of the bloodvessels, and thus allowing a ready swelling of the bronchial mucous membrane. Sanderson ascribes the paroxysm to a dependency on a change in the respiratory function of the vocal cords. He believes this to be an extreme relaxation of the vocal cords during slumber, and a narrowing of the rima glottidis. Beau considers asthma to be a bronchial catarrh with viscid secretions. Leyden ascribes the attack to be due to the presence in the bronchi of a peculiar kind of octahedral crystals produced from white blood-corpuscles, which irritate the pulmonary filaments of the vagus, and provoke the bronchial spasm. Weber considers asthma to be a vasomotor neurosis in which an acute catarrh with a dilatation of the vessels of the bronchial mucous membrane is produced. In this theory he has been followed by Störck, who asserts the existence of tracheal congestion as shown by the laryngoscopic mirror. Lately, Sir Andrew Clark has described a condition of the bronchial mucous membrane simulating the wheals of urticaria on the skin, and has sought to prove this to be a pathological condition of the disorder.

That the theory of spasm of the bronchial muscles is the prevailing one must be admitted, but it stands more in the light of medical tradition than proven fact. The experiments made to prove the contractibility of the bronchial fibres by irritation of the vagus have been conflicting, and the untenability of the theory has been notably disproved, both on clinical and experimental grounds, by Wintrich and Germain

Sée. The fact of the circular fibres being of the unstriped variety, in which the contraction must be slow and gradual, absolutely negatives their coöperation in the sudden and transient narrowing necessary to produce the phenomena of asthma. The office of the bronchial muscles seems more probably to be that of supporting muscles, regulating and supporting the bronchi against atmospheric pressure and undue violence of exertion. The recent experiments of Riegel and Edinger have shown conclusively that irritation of the vagus does not provoke a bronchial spasm, but rather a spasm of the diaphragm; Biermer, the earnest defender of the theory of bronchial contractibility, is forced to say that although stimulation of the vagus may not produce marked contractions of the bronchial muscles, we have not yet found the proper experiments necessary to prove it, and possibly the stimulation must be to the pulmonary sympathetic plexus rather than to the vagus. That spasm of the glottis and spasm of the diaphragm occur in the asthmatic cannot be denied by any one who has made a study of the disorder; the symptoms of those conditions are so marked, and their occurrence so frequent, that they cannot be overlooked; but that they are part of the essential nature of asthma is not true, as we find many cases of asthma in which they are wanting. Spasm of the diaphragm can never produce the mechanical râles always found in asthma, although in certain cases it may play an important part in producing the great distention of lung vesicles seen in the later stage of the disease. The theory of Weber, that the paroxysm is due to vasomotor turgescence or dilatation of the mucous membrane of the bronchi, finds complete support in the clinical picture of asthma: the transient and rapid variations of the physical sounds, the presence of the dry and moist râles, and the acute distention of the lungs seen in pronounced attacks of the disease. This theory, however, is directly contradictory to the acknowledged physiological effect of the drugs which are generally recognized as most useful in allaying the paroxysm; the nitrites, chloral, and morphia are all known as relaxants and anti-spasmodics, and their use could only increase the distress if the dyspnœa was due to a vasomotor dilatation of the vessels of the bronchial mucous membrane. The action of chloral and morphine could be explained by their effect on the nerve-centres; but the efficacy of the nitrites finds here no support. The physiological action of venous blood and carbonic acid on the arterioles and their actual effect in asthma negative the existence of a vasomotor dilatation of the arterioles as a cause of the paroxysm. Ludwig and Severini have shown that venous blood, when circulated for a length of time, causes the vessels to dilate, and that a large amount of carbonic acid in the blood causes a dilatation of the vessels. The natural sequence of an asthmatic paroxysm is extreme venosity of the blood, with an accumulation of carbonic acid through its imperfect aëration. These conditions in reality existing to such an extreme degree in the asthmatic paroxysm, would simply increase the paroxysm if the disturbance were due to vasomotor paralysis of the vessels; whilst, in fact, we find them to be essential elements in the natural cessation of the attack.

In a paper read before the American Laryngological Society, at Detroit, in 1885, I described a condition of the nasal mucous membrane seen in certain patients. This condition is characterized by pallor, swelling, and an œdematous condition of the membrane. When irritated as by the touch of a probe or any other irritant, the swelling is increased and a profuse, thin or watery secretion is poured out. This condition promptly subsides after the inhalation of nitrite of amyl, ether, or the instillation of atropine or morphia; but remains unchanged on the application of cocaine. These cases occurred in persons of a marked nervo-vascular temperament, and in whom functional nerve disturbances were common. The existence of pallor, more or less marked, and a marked œdematous condition of the mucous membrane, distinguish this condition from that ordinarily seen in vasomotor catarrh. In the latter condition there is hyperæmia with dilatation of the vessels; but in the former the pallor completely negatives the idea of a dilated condition of the vessels, and the opposite condition must exist. The action of the drugs indicated in these cases rather a spasm of the arterioles, and I have chosen to consider the disturbance of the nasal membrane to be due to a vasomotor spasm of the arterioles of the membrane. The following case, in which this peculiar condition was the accompaniment of a facial neuralgia, is presented as a perfect type of the condition:

A. M., merchant, florid, stout, about forty-five years of age, was brought to me, complaining of facial neuralgia of the left side and great obstruction in the nostril of that side. He said that this obstruction of the nostril had occurred simultaneously with the attack of the neuralgia; that at times it was so great as completely to close the canal. There was a profuse discharge of a thin, limpid fluid, which was greatly increased on the application of any irritant, such as dust, or the touch of the probe. These same agents also had a marked effect in increasing the swelling of the cavernous tissue. On inspection the right nostril was found normal; the left nostril was almost completely occluded by the pallid membrane of the swollen cavernous bodies, almost as pale as that seen in a corpse. It presented an œdematous, boggy appearance, as if the whole tissue was saturated with secretion, and on any irritation this was poured out in a profuse discharge of a thin, limpid character. This entirely subsided with a subsidence of the facial neuralgia.

A study of these cases led me to surmise that possibly an analogous condition may exist in the mucous membrane of the bronchi during asthmatic attacks. I examined a large number of asthmatics during the paroxysm with the laryngoscope mirror, and found that during the paroxysms the mucous membrane of the pharynx, including the uvula, presented a similar pallid, œdematous appearance; that the membranes of the larynx and trachea appeared paler and more swollen than normal, but not so œdematous as that of the pharynx. When portions of the membrane were inflamed, as is common in asthma, the inflamed area presented more the pale red of an intensely anæmic or phthisical subject than the rosy red of ordinary hyperæmia or inflammation. This is in direct contradiction to the observation of Störck, who claims to have found the larynx and trachea congested during the attack. If these conditions can be seen to exist during the asthmatic paroxysms in the upper air-passages as far as inspection is possible, I think we are fully justifiable in reasoning that an analogous condition exists in the bronchi. The normal functional differences between the mucous membrane of the bronchi and that of the upper air-passages would somewhat change the picture, more especially as the excessive glandular secretions would be greatly diminished in quantity or even wanting. A similar condition to this we find existing in the sympathico-tonica type of migraine (Eulenburg), with vascular spasm and diminished supply of blood in the brain.

I would, then, consider asthma to be a disorder of vascular irritability; that the paroxysm is directly due to a partial occlusion or cylindrical narrowing of the lumen of the bronchi through the swelling of the bronchial mucous membrane; that this swelling is caused by a vasomotor spasm of the arterioles with a saturation of the tissues by the liquor sanguinis; this condition is accompanied by a general high blood pressure.

With this theory we find a complete explanation of all the physical signs of asthma. We see the possibility of the rapid changes of physical signs observed during the paroxysms, and we have in its support the physiological action of all the drugs which experience proves to be of value in allaying the paroxysm—the nitrite of amyl, morphia, chloral, lobelia, and iodide of potash in their action allay the spasm at the same time that they tend to reduce general blood pressure; whilst drugs like the bromides prove of little value in breaking the paroxysms when once in force, although they are serviceable in preventing a recurrence.

ENVIRONMENT IN ITS RELATION TO THE PROGRESS OF BACTERIAL INVASION IN TUBERCULOSIS.

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Though much light has been thrown of late on the etiology of tuberculosis by the labors of Koch, Baumgarten, Weigert, and others, the conditions which lead to tubercular infection are yet shrouded in